

CORRESPONDENCE

Tension pneumothorax case report is misleading

The problem with the recent case report 'Tension pneumothorax mimicking giant emphysematous bullae'¹ is that the patient clearly did not have a tension pneumothorax. He was not particularly unwell and had only a minimal mediastinal shift. A much greater mediastinal shift can be seen in non-tension pneumothorax.² The actual definition of tension pneumothorax is not generally agreed. The 2003 British Thoracic Society guidelines³ refer to tension pneumothorax occurring 'when the intrapleural pressure exceeds the atmospheric pressure throughout inspiration as well as expiration'. By 2010, this had been modified to 'exceeds atmospheric pressure for much of the respiratory cycle'.⁴ The classical division of pneumothoraces into open, closed and valvular has no evidence to support it. It seems extremely unlikely that in so-called open pneumothoraces that air in the pleura actually re-enters the collapsed lung on expiration and is breathed out and we do have experimental evidence that this does not occur. Even supposing the putative valve mechanism existed, the laws of physics preclude pressures during inspiration from being above atmospheric pressure.⁵ In the expiratory phase, pressures in all pneumothoraces exceed atmospheric pressure if there is anything but totally passive expiration, otherwise intercostal drainage of pneumothoraces without suction would not work. It is for this reason that the much taught 'hiss' sign is worthless.

Published reports of tension pneumothorax in spontaneously breathing patients are very rare and none are convincing.² Some, as in the case reported by Gonzalez *et al.*, are healthy patients misdiagnosed on radiological appearances and the rest are patients with severe trauma and large pneumothoraces who unsurprisingly tolerate the subsequent hypoxaemia badly. Hypoxaemia rather than cardiovascular collapse is in fact the consistent finding in experimental pneumothorax in animal models. Severe consequences of this with rapidly expanding pneumothoraces in trauma patients or those with lung disease are not unexpected but the mechanism is not the generation of supra-atmospheric pressure. The term tension pneumothorax should be abandoned for spontaneously breathing patients and reserved for those undergoing positive pressure ventilation.

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Authors' response

We thank Dr Simpson for his comments on our recently published paper entitled 'Tension pneumothorax mimicking giant emphysematous bullae'.¹ Based on the literature, Dr Simpson states the use of the terminology 'tension pneumothorax' in our study as being inaccurate. He suggests that it should only be applied to positive pressure ventilated patients as only they can develop supra-atmospheric intrapleural pressure during much of the respiratory cycle.

We agree that 'tension pneumothorax' is an overquoted terminology in clinics if we strictly consider its physiological definition (supra-atmospheric pressure for much of the respiratory cycle).² It is, however, generally used as a synonym for 'expanding pneumothorax' which is far more common and which, we believe, occurred in the case we report.¹

Some studies consider the one-way valve theory as the most plausible explanation of 'expanding pneumothorax'.² In this model, air can enter the pleural space through a pleural breach and build up pressure especially during the expiratory phase of the respiratory cycle. This is mostly responsible for lung collapse leading to blood shunt phenomena which ultimately cause hypoxia. In rare cases, non ventilated patients can experience a build up of their pleural pressure which can shift their mediastinum and affect cardiac function (return and output).³ This last phenomenon is far more likely in patients undergoing positive pressure ventilation as air is insufflated under supra-atmospheric pressures.

In the patient we report on, the major symptom was progressive dyspnoea with no haemodynamic instability. First, we believe that progressive dyspnoea over a short period of time is compatible with an expanding pneumothorax. Second, the mediastinal shift observed fits well with this picture, although it has also been reported in the absence of expanding pneumothoraces. Third, the patient was known to have severe chronic obstructive pulmonary disease and thus

limited reserves. Altogether, pleural decompression was indicated and caused an important hiss of air during the drain placement with immediate improvement of symptoms.

When managing pneumothoraces, we believe that the most difficult challenge is to appropriately identify patients who require drainage. We have read with great interest Dr Simpson's paper on observational management of spontaneous pneumothoraces.⁴ While we believe this approach is a good alternative to drainage in young patients who develop spontaneous pneumothoraces, we do not think it is applicable to many situations. To our knowledge, any patient with a diagnosed pneumothorax who experiences a progression of symptoms, such as dyspnoea, should be promptly drained, especially if his lung functional reserve is limited. In these particular situations, a one-way valve mechanism can be suspected. In awake patients, this mechanism will never result in an intrapleural pressure that exceeds the atmospheric pressure during the inspiratory phase, which can occur in ventilated patients and cause cardiac collapse. Thus, expanding and tension pneumothoraces are similar in their pathophysiology but have different extremes of the intrapleural pressure that can develop.²

In conclusion, from a pure pathophysiological point of view, our case report title should have been entitled 'Expanding pneumothorax mimicking giant emphysematous bullae'. However, conceptwise, the mechanism of expanding pneumothorax is comparable with tension pneumothorax and the treatment is identical.

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